IN THE SPECIFICATION

Please replace the text beginning at page 14, line 20 through page 15, line 8, with the following text:

Alternatively, a furin deficient cell may be obtained by exposing cultured cells to mutagenesis treatment, e.g., irradiation, ethidium bromide, bromidated uridine (BrdU) and others, preferably chemical mutagenesis, and more preferred ethyl methane sulfonate mutagenesis, recovering the cells which survive the treatment and selecting for those cells which are found to be resistant to the toxicity of *Pseudomonas* ~~exodotoxin~~ exotoxin A (see Moehring and Moehring (1983) Infection and Immunity 41(3):998-1009).

The amount of *Pseudomonas* ~~exodotoxin~~ exotoxin A can be used as described *supra*, or can be empirically determined for each individual cell type by titrating various concentration of *Pseudomonas* ~~exodotoxin~~ exotoxin A on the cells and observing the concentration of *Pseudomonas* ~~exodotoxin~~ exotoxin A, which does not result in the killing of all the cells. A preferred range includes 0.5 to 2.0 μg/ml, including 0.75, 1.0, 1.25, 1.5, 1.75, and all values therebetween.

Please replace the text beginning at page 16, line 9 through page 17, line 10, with the following rewritten text:

In another embodiment of the invention, the cells found to be furin deficient may also be subsequently or previously selected for lectin resistance ~~resitance~~, preferably ricin resistance as described in Applicants co-pending U.S. applications: "METHOD OF PRODUCING GLYCOPROTEINS HAVING REDUCED COMPLEX CARBOHYDRATES IN MAMMALIAN CELLS" U.S. application serial no10/023,890, which was filed 12/21/01or METHODS OF PRODUCING HIGH MANNOSE GLYCOPROTEINS IN COMPLEX CARBOHYDRATE DEFICIENT CELLS", U.S. application serial no

10/023,889, which was filed 12/21/01 the contents of which are incorporated herein by reference.

Any lysosomal enzyme that uses the M6P transport system can be treated according to the method of the present invention. Examples include α-glucosidase (Pompe Disease), α–L-iduronidase (Hurler Syndrome), ~~β-galactosidase A~~ α-galactosidase A (Fabry Disease), arylsulfatase (Maroteaux-Lamy Syndrome), N-acetylgalactosamine-6-sulfatase or β-galactosidase (Morquio Syndrome), iduronate 2-sulfatase (Hunter Syndrome), ceramidase (Farber Disease), galactocerebrosidase (Krabbe Disease), β -glucuronidase (Sly Syndrome), Heparan N-sulfatase (Sanfilippo A), N-Acetyl-α-glucosaminidase (Sanfilippo B), Acetyl CoA-α-glucosaminide N-acetyl transferase, N-acetyl-glucosamine-6 sulfatase (Sanfilippo D), Galactose 6-sulfatase (Morquio A), Arylsulfatase A, B, and C (Multiple Sulfatase Deficiency), Arylsulfatase A Cerebroside (Metachromatic Leukodystrophy), Ganglioside sialidase (Mucolipidosis IV), Acid β-galactosidase G_{M1} Galglioside (G_{M1} Gangliosidosis), Acid β-galactosidase (Galactosialidosis), Hexosaminidase A (Tay-Sachs and Variants), Hexosaminidase B (Sandhoff), α-fucosidase (Fucsidosis), α-N-Acetyl galactosaminidase (Schindler Disease), Glycoprotein Neuraminidase (Sialidosis), Aspartylglucosamine amidase (Aspartylglucosaminuria), Acid Lipase (Wolman Disease), Acid Ceramidase (Farber Lipogranulomatosis), Lysosomal Sphingomyelinase and other Sphingomyelinase (Nieman-Pick).